University of Minnesota
The University Library
Minneapolis 14, Minn.

UNIVERSITY OF MINNESOTA
RECEIVED
JUL
5
1951
LIBRARY

CLINICAL PROCEEDINGS

of the

BIO-MEDICAL LIBRARY

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

SAMPLE COPY

May 1951

VOLUME VII NUMBER 6

PEOPLES DRUG STORES
INCORPORATED
AND SUBSIDIARY CORPORATIONS

WASHINGTON 2, D. C.

TO THE PROFESSION:

We carry in stock the drugs you prescribe; whether they be proprietary, trade named, official, or ingredients to be compounded to your order. New drugs are added as soon as the manufacturer makes them available.

We demand quality merchandise from all manufacturers, playing no favorites in prescription stocks. We realize that the medical service representative of any reputable manufacturer, who creates a demand in the profession for an item, deserves credit for the prescription. Only the item specified may be dispensed.

You are assured your patients will receive what you write for when they bring their prescriptions to Peoples.

Very truly yours,

PEOPLES DRUG STORES, INC.

Raymond Watson

Raymond Watson
Professional Service Department

CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

13th and W Streets, Washington 9, D. C.

Vol. VII *May 1951* No. 6

CONTENTS

THE UROGENITAL ANOMALIES. A NECROPSIED SERIES. *Charles L. Waite, M.D.; Joseph M. LoPresti, M.D.; Preston A. McLendon, M.D.; E. Clarence Rice, M.D.; Hilbert S. Sabin, M.D., and John E. Cassidy, M.D.*.... 145

ESOPHAGEAL ATRESIA WITH TRACHEO-ESOPHAGEAL FISTULA. *Marshall C. Sanford, M.D.*..... 151

CLINICO-PATHOLOGICAL CONFERENCE. *Richard J. Waters, M.D.; and Bernice Weedum, M.D.*..... 166

EDITOR-IN-CHIEF

E. CLARENCE RICE, M.D.

MANAGING EDITORS

FREDERIC G. BURKE, M.D. SIDNEY ROSS, M.D.

ASSOCIATE EDITORS

JOSEPH M. LOPRESTI, M.D. JOHN P. MCGOVERN, M.D.
ADRIAN RECINOS, JR., M.D. MARSHALL C. SANFORD, M.D.

GENERAL MANAGER

JEANNE DECHANTAL RODDY

Contributing Editors from the Resident Staff: WARREN G. PREISSER, M.D., ROBERT ANDERSON, M.D., DAVID F. BELL, M.D., GORDON W. DAISLEY, M.D., SANFORD LEIKIN, M.D., ELLERY MICHAUD, M.D., ROBERT PARROTT, M.D., WILLIAM L. SAGER, M.D., AND RICHARD J. WATERS, M.D.

Publications Committee of the Medical Staff: E. CLARENCE RICE, M.D., THOMAS BRADLEY, M.D., WILLIAM A. HOWARD, M.D., FREDERIC G. BURKE, M.D., AND SIDNEY ROSS, M.D.

Publications Board of the Board of Directors of Children's Hospital: ORME WILSON, WILSON B. NAIRN, SAMUEL SPENCER, WALLACE WERBLE, E. CLARENCE RICE, M.D., FREDERIC G. BURKE, M.D., AND MONTGOMERY BLAIR, JR., M.D.

Published monthly by the Staff. Cases are selected from the weekly conferences held each Sunday morning at 11:00 A.M., from the Clinico-pathological conferences held every other Tuesday afternoon at 1:00 P.M., and from the monthly Staff meetings.

This bulletin is printed for the benefit of the present and former members of the Attending and Resident Staffs, and the clinical clerks of Georgetown and George Washington Universities.

Subscription rate is $1.00 per year. Those interested make checks payable to "Clinical Proceedings Dept.," The Children's Hospital, Washington, D. C. Please notify on change of address.

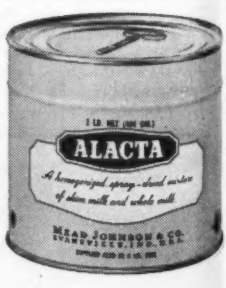
Copyright 1951, Children's Hospital

Entered as second class matter November 21, 1946 at the post office at Washington, D. C., under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 538, Act of February 28, 1925, authorized January 17, 1947.

ALACTA® is a *high protein, low fat* powdered milk designed to meet the special nutritional needs of premature infants—those babies who arrive "too little and too soon."

Alacta is valuable for *any* infant for whom physicians desire a high protein, low fat formula. Alacta–Dextri-Maltose® formulas are ideal for babies whose fat tolerance is low, or where gastric emptying time is prolonged as in hot weather or during bouts of infectious disease. Infants suffering from protein deficiency thrive on Alacta–Dextri-Maltose formulas prescribed in accordance with the clinical indications.

Literature (including reprints of studies in which Alacta was employed) and formula tables are available on request.



MEAD'S

MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U. S. A.

THE UROGENITAL ANOMALIES

A NECROPSIED SERIES

Special Report No. 207

Charles L. Waite, M.D.
Joseph M. LoPresti, M.D.
Preston A. McLendon, M.D.
E. Clarence Rice, M.D.
Hilbert S. Sabin, M.D.
John E. Cassidy, M.D.

It is occasionally of value to reemphasize well-known medical facts. Helmholz[1] in 1927 stressed the importance of a complete urinalysis in pediatric practice and the significance of pyuria. Since that time it has become an almost universal practice to perform routine urinalyses on all hospital admissions. Unfortunately, since the urinalysis has become so routine a procedure, abnormal urinary findings are often overlooked. This, together with the abstruse symptomatology produced by obstructive uropathy, often results in delayed recognition of the pathology. Because of these facts, it was felt that a review of the necropsy records at Children's Hospital and of the literature on this subject would be worthwhile. From a practical standpoint this survey was conducted with definite objectives: 1. To ascertain the relative incidence of urogenital anomalies particularly with reference to those which are the primary cause of death. 2. To attempt, if possible, a correlation between the pathology and the clinical symptomatology. In this way, it was felt that clues leading to an early diagnosis of the surgically amenable urogenital anomalies would be uncovered.

A survey of the autopsies performed at this hospital during the 17-year period from 1932 to 1949 was made. In this period, 2,850 post-mortem examinations were done (Table 1). Of this total, 4.7 per cent or 135 patients were found to have anomalies of the urogenital tract. This figure is in accord with numerous other reports in which 5 to 10 per cent of autopsied cases were found to have similar anomalies[1, 2, 3, 4, 5]. The anomalous conditions were divided into those which did not cause the patient's death and those anomalies which were fatal to the patient. The ratio of fatal to non-fatal anomalies was one to four. The fatal developmental defects comprise 1.1 per cent or 31 of the autopsied patients.

The age, sex and racial distribution of these congenital lesions are shown in Table 2. The ages of this group of patients ranged between one week and 12 years with an average of 20 months. Urogenital anomalies were

three times more common in males and over four times as common in white, than in colored, patients. This latter preponderance is not significant since the white admissions at this hospital outnumber the colored admissions by 3 to 1.

Most of the anomalies were not the primary cause of death (Table 3). The majority of these were asymptomatic and physical examination usually indicated the site of pathology. They pose no great problem in management and, for the most part, are included for their statistical value. In the order of occurrence there were undescended testes, duplication of ureters and renal pelves, phimoses, hypospadius, ectopic kidney, hydrocele, and horseshoe kidney. Of interest were 24 children who had some

TABLE 1

Incidence of Urogenital Anomalies in 2,850 Autopsies

Number of Autopsies (1932–1949)	2,850
Number of Urogenital Anomalies	135 (4.7%)
Non-Fatal Anomalies	104 (3.6)%
Fatal Anomalies	31 (1.1%)

TABLE 2

Age, Sex and Racial Distribution in 135 Patients with Urogenital Anomalies

Age range	1 week to 12 years
Average age	20 months
Sex,	
Males	100 (74%)
Females	35 (26%)
Race,	
White	112 (83%)
Colored	23 (17%)

degree of bilateral hydronephrosis and hydroureter. In this group, renal symptoms were entirely absent. At necropsy no cause was found to account for the dilatation. The fatal outcome was ascribed to an unrelated disease. Of necessity these findings must be classified as idiopathic.

By contrast, the more important but less common urogenital anomalies are those which imply a serious prognosis. They were the primary cause of death in 31 children (Table 4). Obviously such entities as renal agenesis and bilateral cystic kidneys signify a hopeless prognosis. With better surgical techniques, effective antibiotics and chemotherapy, the outlook for patients with extrophy of the bladder has improved. Eighteen in this group of 31 patients had a congenital obstruction to the free flow of urine. This obstruction produced a sequence of events which eventually terminated in the patient's death.

Where one anomaly is found in a patient, then other congenital defects

are apt to be present (Table 5). This seems to be particularly true of the fatal urogenital anomalies. In this series 14 or 45.1 per cent had associated anomalous conditions. Spina bifida and developmental defects of the heart were the most common associated anomalies. An imperforate anus was found in two patients.

It is evident that the majority of the fatal anomalies are those which obstruct the urinary tract. If recognized early, these are amenable to sur-

TABLE 3

Distribution of 104 Non-Fatal Anomalies

Idiopathic hydronephrosis and hydroureter	24
Undescended testes	22
Duplication of ureters and kidneys	10
Phimosis	8
Hypospadius	8
Ectopic kidney	7
Hydrocele	7
Horsehoe kidney	6
Pseudohermaphrodism	2
Aberrant renal vessels	2
Unicornuate uterus	1
Total	104

TABLE 4

Distribution of 35 Fatal Anomalies

Uretero-vesicle obstruction	9
Renal agenesis	5
Extrophy of the bladder	5
Polycystic kidney	3
Posterior urethral valves	3
Kink of ureter	3
Uretero-pelvic obstruction	2
Congenital stricture of the urethra	1
Total	35

gical repair. Thirteen of the eighteen congenital obstructions were located at or above the uretero-vesicle openings (Table 6). Of these, nine were due to a bilateral constriction of the uretero-vesicle ostia, two were caused by the pressure of an aberrant blood vessel at the uretero-pelvic junction, and in three patients the obstruction was produced by a kink of the ureter. In the remaining four patients, the point of obstruction was located in the urethra. In three of these, a posterior urethral valve was the original site of obstruction. The fourth had a congenital stricture of the urethra. Although there is a similarity in the sequence of events, there is a difference

in the rate of progression of the uropathy between obstructions located proximal or distal to the uretero-vesicle orifices. When the lesion is at or proximal to this point the course of events is rapid. The course is more insidious and gradual when the lesion is located distally.

The initial pathology is an obstructive focus which creates an impediment to the egress of urine. Proximal to the obstruction, urinary stasis inevitably results. This stagnant pooling of urine provides an excellent culture medium for bacterial growth. *Invariably a pyuria appears.* As urine collects proximal to the obstruction, the hydrostatic pressure increases. All of the proximal structures eventually become dilated. Finally, the terminal

TABLE 5

Congenital Defects Associated with 31 Fatal Urogenital Anomalies

Spina bifida	5
Congenital defect of the heart	4
Imperforate anus	2
Microcephaly	1
Accessory spleen	1
Accessory liver lobe	1
Total	14 (45.1%)

TABLE 6

Sites of Obstructive Lesions in 18 patients

1. At or Above the Uretero-Vesicle Ostia	
Aberrant vessel	2
Kink of ureter	3
Uretero-vesicle constriction	9
2. Below the Uretero-Vesicle Ostia	
Posterior urethral valves	3
Congenital urethral stricture	1

features in obstructive uropathy appear. The constant back pressure of urine ultimately produces necrosis of the renal parenchyma. A progressive renal insufficiency accrues. These changes rapidly become irreversible and result in death.

The early recognition of a congenital obstruction in the urinary tract is imperative. Surgical repair is beneficial only if irreversible renal changes have not occurred. Initially, an alert pediatrician may elicit a history of straining on urination or of large amounts of urine associated with infrequent diaper changes. When infection is present, frequency of urination is a more common complaint. Often, this early symptomatology is overlooked or is unnoticed by the parents. Gold and Jones[6] in an excellent survey of 31 patients with proven hydronephrosis demonstrated that an average of

one year elapsed between the onset of symptoms and the establishment of the diagnosis. In their patients, less than one-half had definite urinary complaints. The occurrence of pyuria in a child should serve as a danger signal to the practitioner. Pool and Cook[7] have emphasized that one of the cardinal features of urinary tract obstruction is recurrent bouts of pyuria. The obstruction must be cared for before the infection can be eradicated permanently. *Whenever a pediatric patient has repeated infections of the urinary tract it is mandatory that intensive efforts be made to locate a possible obstructive focus.* Abdominal pain is an inconstant manifestation. Ladd and Gross[8] affirm that a kidney pelvis may attain enormous proportions and be unaccompanied by pain in its dilated state. The clinical correlation to this stage may be the presence of a palpable intra-abdominal mass. In almost one third of the patients with hydronephrosis, a palpable intra-abdominal mass is the presenting sign[6]. Ultimately the symptoms and signs of irreversible renal damage appear. The non-protein nitrogen of the blood will be elevated in about one-third of the patients before the condition is recognized. This confirms the fact that the diagnosis is too often made late in the disease. Uremia with its skin manifestations, irritability, stupor, coma and convulsions is the final expression of obstructive uropathy. Occasionally one of these patients will exhibit the signs of renal rickets. In this clinical entity an obstruction in the urinary tract is the most frequent instigating factor. When renal insufficiency is chronic, large amounts of calcium are lost in the urine. As calcium is decreased the blood phosphorous becomes elevated. This elevation in circulating phosphorous stimulates the parathyroid glands and they hypertrophy. The resulting secondary hyperparathyroidism produces a further mobilization of calcium from the osseous system. In addition, phosphates are secreted into the intestinal tract in large amounts. Any calcium which is ingested combines in the intestine with these phosphates to form insoluble calcium phosphate. This deprives the patient of his normal exogenous source of calcium, an additional factor in the production of renal rickets. In the early stages of renal rickets there is an equilibrium between calcium loss in the urine and calcium mobilization from the skeletal system. These patients, therefore, have a normal blood calcium and a markedly elevated blood phosphorous. Renal rickets is the only condition which will produce these chemical changes. Terminally, excessive amounts of calcium are lost and the patient dies in tetany. The X-ray picture in these rare patients is similar to the one seen in rickets. The histologic bone changes are those of osteitis fibrosa cystica.

The clinical picture presented by children with congenital defects of the urinary tract is obviously a deceptive one. The early symptoms direct attention to the site of pathology in less than half of the patients. The

prognosis becomes more unfavorable with lapse of time, and when irreversible renal damage has occurred the situation is nigh hopeless. Early diagnosis and treatment are essential for a favorable outcome. Success is directly related to the cognizance of these facts by the physician. In the presence of a pyuria the pediatrician should sense the possibility of more serious pathology. It is imperative that intensive efforts be made to locate a possible obstructive focus in any pediatric patient who has repeated bouts of pyuria, if the obstructive lesions in the urinary tract are to be recognized in their initial stages.

SUMMARY

1. 2,850 autopsies were performed at the Children's Hospital between the years of 1932 and 1949, and 135 of these had anomalies of the urogenital tract for an incidence of 4.7 per cent. They were the cause of death in 31 patients.

2. The urogenital anomalies were noted to be three times more common in males.

3. The most common fatal developmental defects were found to be those which obstruct the urinary tract.

4. The pathology, symptomatology and recognition of obstructive uropathy have been summarized. Often an early diagnosis will be indicated by a complete urinalysis. Repeated attacks of pyuria make intensive investigation of the urinary tract mandatory.

5. Finally, awareness of the presented facts by the pediatrician is the key to success in early diagnosis and treatment of obstructive uropathy.

BIBLIOGRAPHY

1. Helmholz, H.: Congenital Abnormalities of the Urinary Tract in Childhood. J. A. M. A. **89:** 1932, 1927.
2. Bigler, J. A.: Anomalies of the Urinary Tract in Children: Frequency and Types in a Consecutive Necropsy Series. Am. J. Dis. Child. **38:** 960, 1929.
3. Bugbee, E. P. and Wollstein, M.: Surgical Pathology of the Urinary Tract in Infants. J. A. M. A. **83:** 1887, 1924.
4. Campbell, M. F.: Pediatric Urology. Macmillan, New York, 1937.
5. Kretschmer, H. L.: Genito-Urinary Conditions in Infants and Children. J. Peds. **30:** 603, 1947.
6. Gold, P. and Jones, J. P.: Hydronephrosis; A Case Report and a Review of 31 Cases. Clin. Proc. Child. Hosp. **4:** 1, 38, 1948.
7. Pool, T. L. and Cook, E. N.: Present Concepts of Treatment of Infections of the Urinary Tract. J. A. M. A. **133:** 584, 1947.
8. Ladd, W. E. and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, W. B. Saunders, Philadelphia, 1941.

ESOPHAGEAL ATRESIA WITH TRACHEO-ESOPHAGEAL FISTULA

Special Report No. 208

Marshall C. Sanford, M.D.

Esophageal atresia with tracheo-esophageal fistula is a congenital anomaly which is incompatible with life unless corrected early. The deformity usually consists of a complete atresia of the upper esophagus which ends blindly at the level of the third or fourth thoracic vertebra. The distal esophagus in most cases communicates with the trachea at or a little above the carina.

Turner[1] states that this anomaly occurs about once in every 2,500 births or with about the same frequency as harelip and cleft palate. Recently Belsey and Donnison[2] suggested that more of these cases are misdiagnosed than was previously assumed and that the actual incidence is about 1 case in about every 800 births. This condition was described by Thomas Gibson[3] of London in 1703, but it was not until 1939 that the first successful repairs were reported by Leven[4] and by Ladd.[5] These patients were treated by exteriorization of the proximal esophagus, closure of the fistula, and gastrostomy. At a later date, continuity of the intestinal tract was established by multiple-stage ante-thoracic esophagoplasty. It remained for Cameron Height to perform the first successful primary end-to-end anastamosis which he reported in 1943.[6] Prior to that time, most of the cases had gone unrecognized, or had been considered hopeless and were allowed to die without operation.

The findings at or shortly after birth are characteristic enough to make the diagnosis easy if the possibility of this deformity is kept in mind. It is so important to make the diagnosis early that active steps to establish this diagnosis should be taken in any case in which it is suspected.

The classification according to Ladd[5, 7] is listed and illustrated below.

Class I. The upper esophageal segment terminates in a blind pouch in its upper one-third and the lower esophageal segment is represented by a similar blind pouch. There is no communication with the trachea.

Class II. The upper esophageal segment communicates with the trachea and the lower segment consists of a blind pouch.

Class III. The upper segment terminates blindly while the lower esophageal segment communicates with the trachea above the carina.

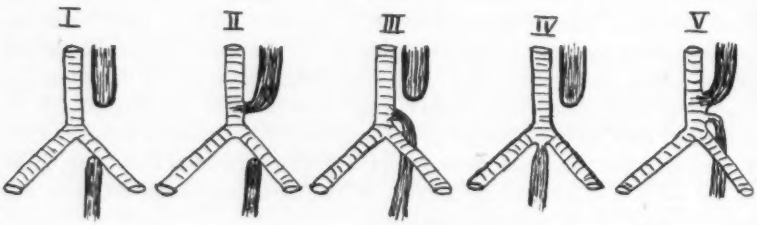
Class IV. Similar to Class III except the lower segment communicates with the trachea at the carina or with one of the main-stem bronchi.

Class V. Both upper and lower esophageal segments communicate with the trachea.

Ninety to ninety-five per cent of all cases fall into groups III or IV which usually are considered together since the anomaly is essentially the same.

Diagnostic Procedures and Pre-Operative Management: Esophageal atresia should be suspected in any newborn infant who seems to have an excessive amount of saliva and mucous in his mouth, and/or who has episodes of coughing and cyanosis, and/or who develops pneumonia or atelectasis during the first few days of life. Excessive mucous in the mouth and throat immediately following delivery should suggest this possibility, and the regurgitation of feedings immediately after swallowing, associated with coughing and cyanosis, is almost pathognomonic. Rarely the first sign will be evidence of pneumonia (due to aspirated saliva or liquids) or atelectasis (due to occluded bronchi). This mucous may be bile-stained due to the presence of the tracheo-esophageal fistula and may be a source of confusion in establishing the diagnosis. Many of these babies are premature and associated anomalies are common.

TABLE 1



The diagnosis is established by:

1. The introduction of a small (No. 8 French) catheter through the nose into the proximal esophageal segment. Usually an obstruction is met at the level of the third or fourth thoracic vertebra. This examination always should be performed under a fluoroscope because it is possible for the catheter to curl back on itself in the blind pouch without giving any sense of resistance during the passage.

2. Aspiration of all mucous and retained secretions from this blind pouch to prevent further spill-over into the lungs.

3. The introduction of 1 to 2 milliliters of lipiodol to outline the lower limit of the blind pouch. The use of barium is contraindicated since it causes a chemical pneumonitis and mechanically blocks off many of the smaller bronchi.

Roentgenographic examination of the chest and abdomen should be done to determine the type and extent of the pulmonary involvement. X-ray is also helpful in detecting the presence or absence of air in the

intestinal tract. The amount and distribution of this air, if it exists, may establish the presence of associated anomalies. If air is present in the stomach and intestines, a fistula between the distal esophageal segment and the trachea exists. However, its absence does not entirely rule out the presence of this fistula since the opening may be extremely small. Haight and Potts have reported cases in which the presence of the fistula was not suspected before operation.

Once the diagnosis is established, the patient should be prepared as rapidly as possible for operation. This period of treatment should be short and efficiently organized so that no time is wasted and the patient is ready for operation within a few hours. Pre-operative management is aimed at

TABLE 2

Anomalies and Complicating Associated Conditions.
Multiple Deformities Common—Some Incompatible with Life

1. Prematurity*
2. Imperforate anus†
3. Pneumonia and/or atelectasis
4. Underweight (prognosis altered by weight)
5. Cardiovascular anomalies
6. Small bowel atresia
7. Subdural hemotomas (unsuspected)
8. G. U. tract anomalies
9. Brain tumor
10. Facial paralysis
11. Cleft palate and hare lip
12. Deformity of ear
13. Hypertrophic pyloric stenosis

*In 35 cases analysed by Potts, 9 weighed less than 5 pounds and these patients all died.

† In 94 infants with tracheo-esophageal fistula reported by Pless, 59 had associated anomalies. Imperforate anus was the anomaly in 24 of these cases.

combating the existing pneumonia, preventing further aspiration, and establishing fluid and electrolyte balance. The following steps have been found beneficial during this pre-operative period.

1. The child is placed in a crib with the head elevated and a catheter inserted into the proximal esophageal segment. This catheter is attached to gentle suction. This position allows the secretions to be aspirated as they accumulate in this blind pouch, and also tends to prevent regurgitation of intestinal secretions through the fistula. This is the single, most important step in the pre-operative management!
2. Nothing is given by mouth.
3. Suitable antibiotics to combat the existing pneumonia.
4. Oxygen should be administered by tent.

5. Constant, special nursing care must be available.

6. Fluid and electrolyte balance should be established and maintained, but *it is extremely important to restrict the intake of salt.* These patients can become edematous and less than the normal amount of fluid should be given. This fluid intake should be recorded and regulated thoroughly. It is wise to give saline only with the blood or plasma.

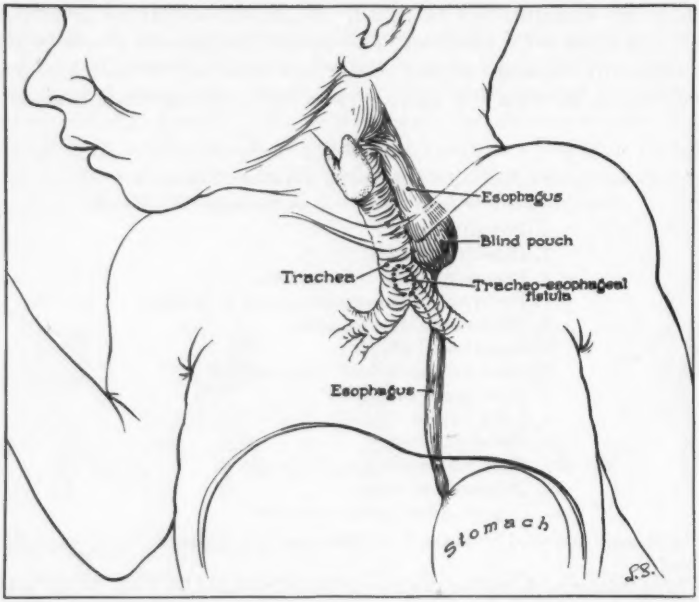


FIG. 1. Drawing of the usual type of anomaly in esophageal atresia. The upper segment thick-walled and dilated, ends blindly. It is often attached to the lower segment by a few muscle fibers. The lower segment, slender and delicate, communicates with the trachea. (*From: Ravitch, M. D., "New Trends in Pediatric Surgery," Surg. Clin. N. A., W. B. Saunders Co. Oct. 1949.*)

7. The baby should be kept on the side to be operated or on the side in which the inflammatory process is most marked, thus tending to limit it to this area.

8. Operation should be performed as soon as the above have been completed since needless delay greatly increases the mortality.

9. Atropine should be given to reduce secretions; and morphine, as preoperative sedation.

Operation: The selection of anesthesia is still a matter of individual

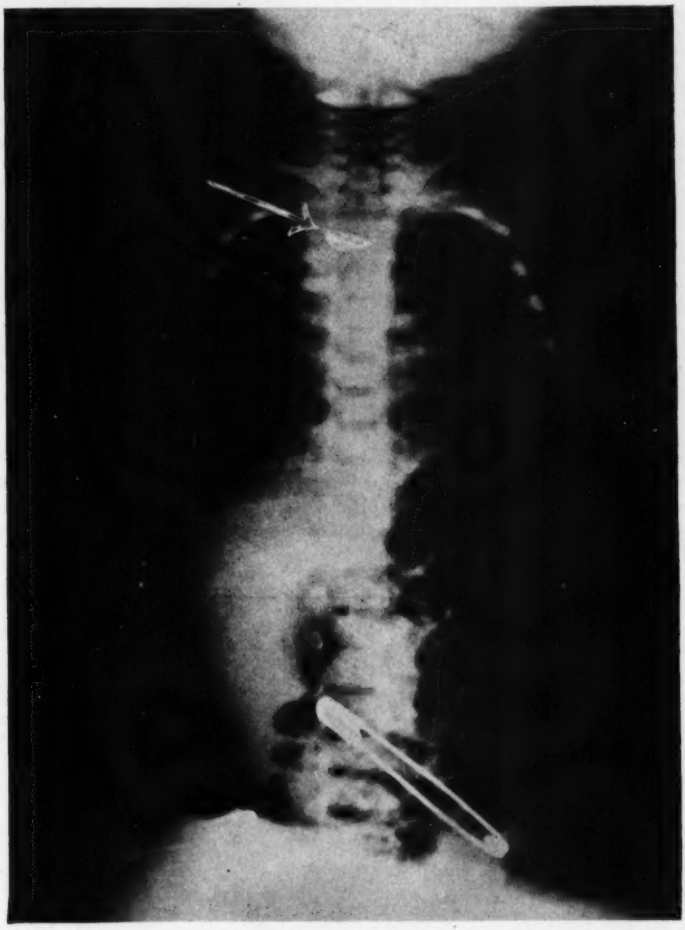


FIG. 2. Roentgenogram of the chest with lipiodol outlining a blind proximal esophageal pouch. Note the distribution of air in the intestinal tract thereby demonstrating the presence of a fistula between the distal esophagus and the tracheal tree.

preference, but most surgeons feel that local anesthesia is not satisfactory and prefer cyclopropane or ether administered through an endo-tracheal tube or a tight-fitting mask. An extrapleural or intrapleural approach on both the right or the left have been utilized, but the one that has proven most satisfactory consists of a right extrapleural approach (the baby lying

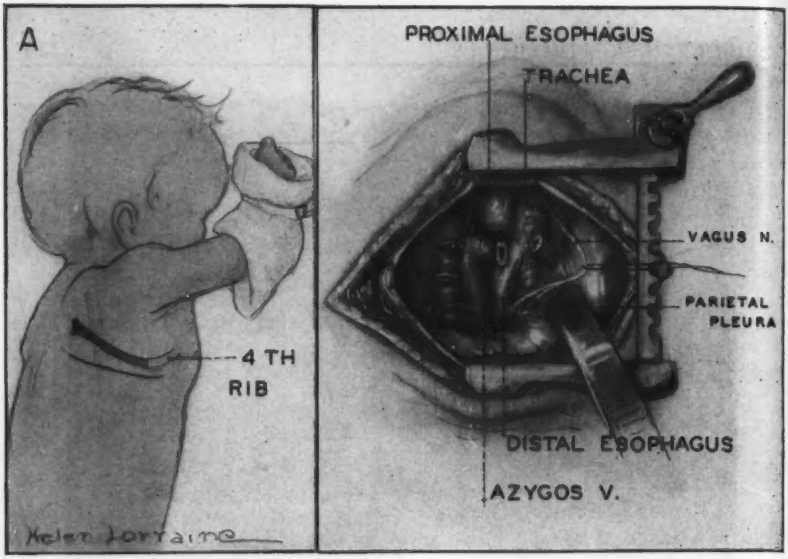


FIG. 3. Excellent exposure is obtained when the major portion of only one rib is removed. The approach is still extrapleural. (*From: Bigger, I. A., "Treatment of Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula," Ann. Surg., J. B. Lippincott Co. May 1949.*)

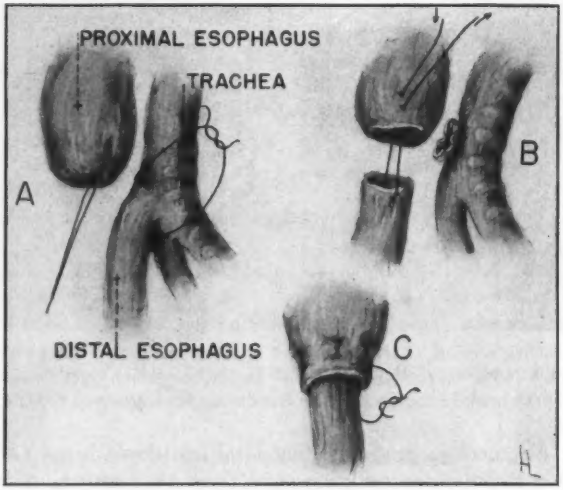


FIG. 4. One method of closing the fistula and performing the anastamosis is demonstrated. The smaller distal esophageal segment may be telescoped into the larger proximal segment. Note the verticle application of the sutures. (*From: Bigger, I. A., "Treatment of Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula," Ann. Surg., J. B. Lippincott Co. May 1949.*)

on his left side) in which either the major portion of the third or fourth rib is resected, or short segments of the second, third, and fourth are removed. A small retractor is introduced and the intercostal muscle bundles are divided if necessary. The lung is gently mobilized anteriorly and medially. The proximal esophageal segment is greatly thickened and dilated due to

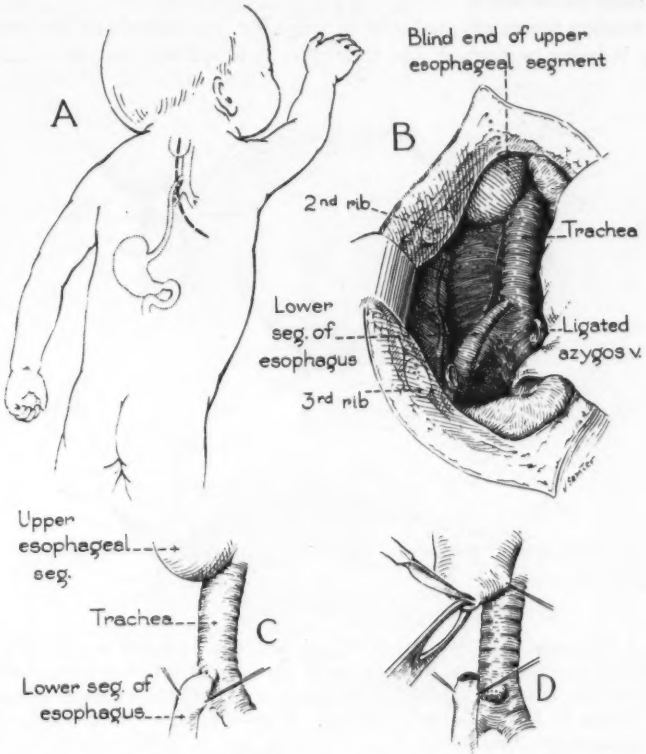


FIG. 5. The drawing illustrates the most commonly employed incision—an extrapleural approach to the esophageal segments and the fistula. Segments of two or more ribs are removed. (*From: Potts, W. J., "Congenital Deformities of the Esophagus," Surg. Clin. N. A., W. B. Saunders Co. February 1951.*)

futile attempts at swallowing during the last weeks of intrauterine life. The distal segment is small and thin-walled. The fistula is identified and closed as soon as possible to prevent regurgitation from the stomach or distension resulting from pumping anesthetic gases into the intestinal tract. The proximal esophageal segment is then freed-up for as great a distance into

the neck as one can reach. It is wise to limit the dissection of the distal segment since its blood supply is not as good. If the two ends can be safely joined together without undue tension, a careful end-to-end anastamosis is performed using interrupted silk sutures. Some workers have suggested telescoping the distal segment into the larger proximal one to insure a more snug anastomosis.

By passing the esophageal tube through the anastamosis at the time the suture is being performed, the procedure is simplified; but once this has

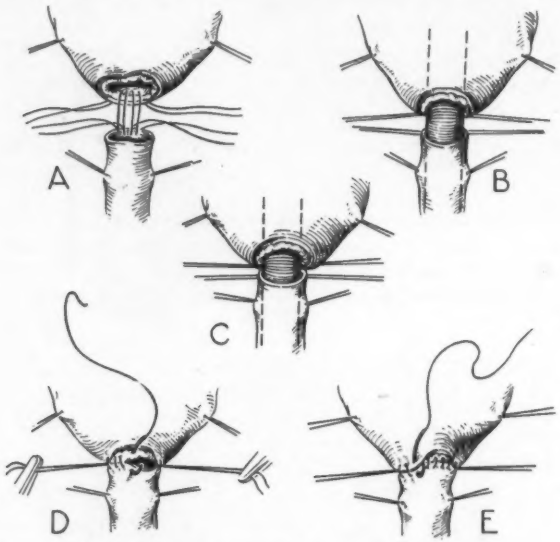


FIG. 6. A careful two-layered, interrupted suture anastamosis is performed. The passage of a tube through the anastamosis at the time the suture is being performed aids in this maneuver. It should be withdrawn once the anastamosis is completed. (From: Potts, W. J., *"Congenital Deformities of the Esophagus" Surg. Clinics N. A., W. B. Saunders Co., Feb. 1951.*)

been completed, it is important to draw the tube back proximal to this suture line.

Before closing the wound, either a small multiperforated catheter or rubber drain should be carried down to the region of the suture line and brought out through a small separate stab wound. If a catheter is used, it is wise to attach this to water-seal suction.

Blood is given during the operation.

A gastrostomy is performed 24 to 48 hours following the primary anastamosis and local anesthesia is quite satisfactory for this procedure. It is

desirable to begin gastrostomy feedings early. A six-hours delay is safe and feedings may be initiated after this time. Bigger[8] has recently advocated a gastrostomy prior to repair of the fistula and esophageal atresia.

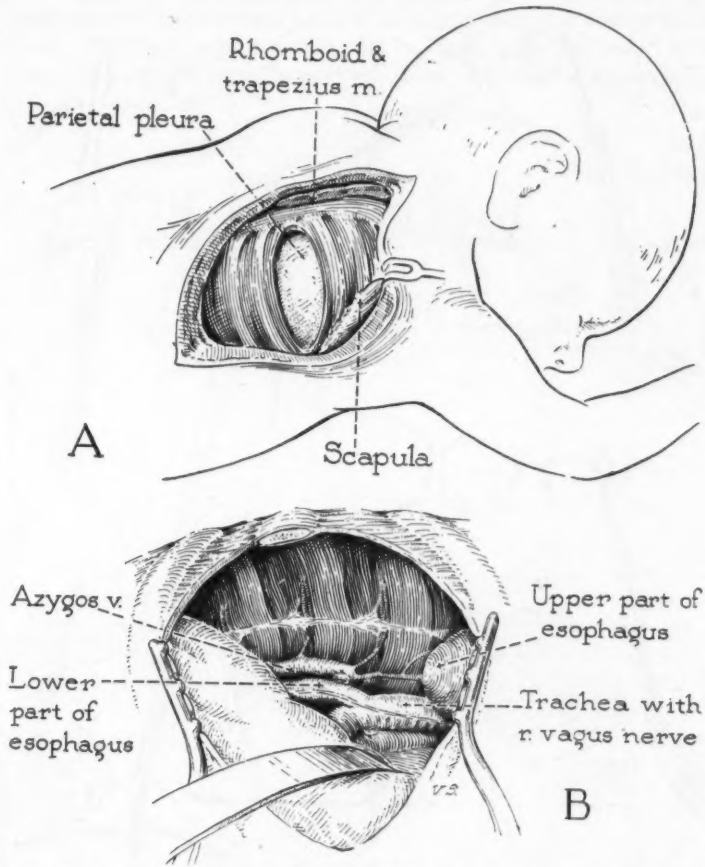


FIG. 7. Extrapleural exposure of the proximal and distal esophageal segments with the tracheo-esophageal fistula easily visible. (*From: Potts, W. J., "Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula," J. Thoracic Surg. November 1950.*)

His reason for this is to deflate the intestinal tract and to prevent regurgitation of intestinal contents into the bronchial tree by employing gentle suction. Obviously, it is extremely dangerous to begin feedings prior to

closure of the fistula and most workers still feel the gastrostomy should follow the primary repair.

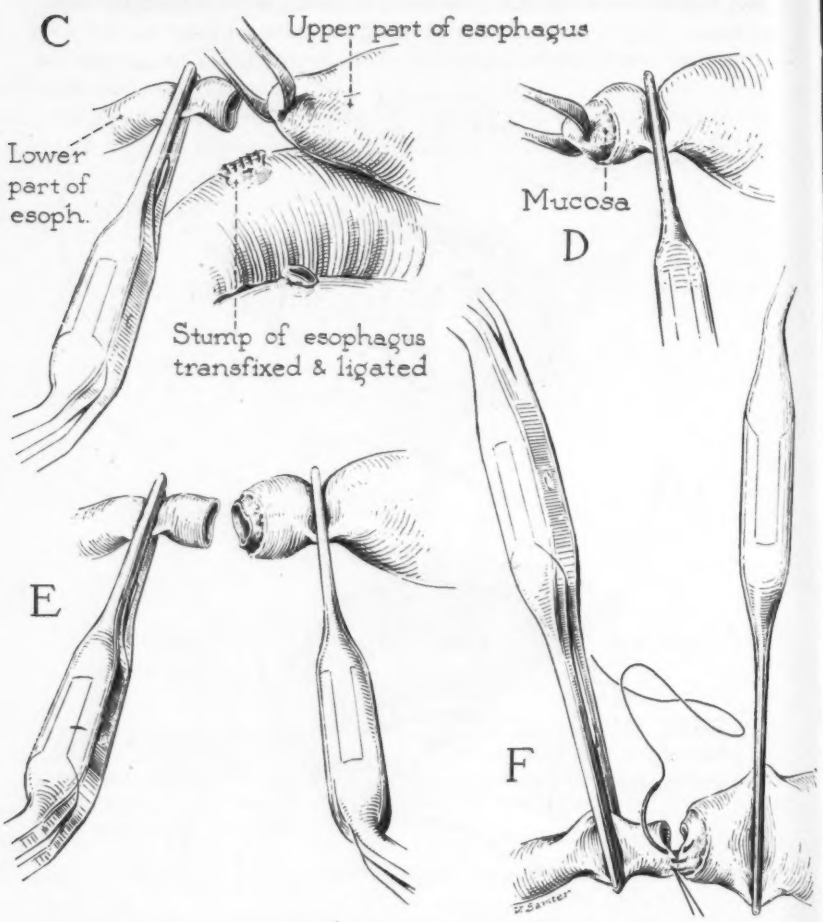


FIG. 8

FIGS. 8 and 9. One method of performing an end-to-end anastamosis utilizing non-crushing, fine-toothed Potts clamps to enable suturing without tension. (*From: Potts, W. J., "Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula," J. Thoracic Surg. November 1950.*)

In a certain number of cases (estimated at about 1 in 15) the two esophageal segments cannot be brought together without undue tension or

in those cases in which agenesis of the distal segment exists, primary union cannot be accomplished. In these the fistula is closed, the proximal esophageal segment is brought out in the neck as a cervical fistula; the distal segment is closed, and a gastrostomy is performed. Continuity of the intestinal tract is reestablished usually at a later date by one of several methods. The jejunum or stomach may be brought up and joined to the cervical-esophageal stump. The multiple-stage, skin tube esophagoplasties

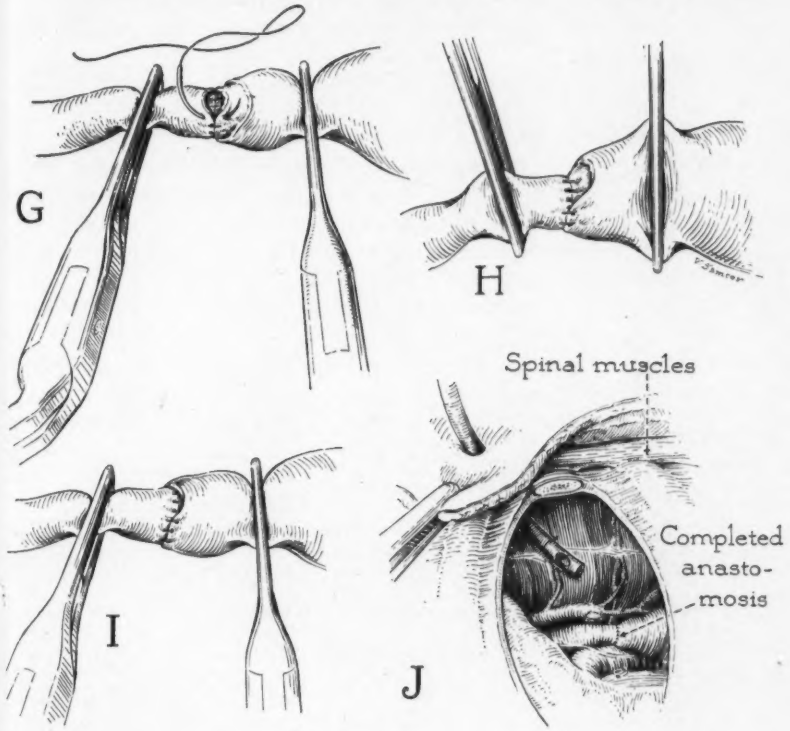


FIG. 9

are to be condemned because of their frequent failures, prolonged operative periods, and poor cosmetic results.

Post-operative management:

1. Administer oxygen by tent.
2. Continue antibiotics.
3. Place catheter in proximal esophageal segment and maintain gentle suction.

4. Combat edema with plasma and albumin.
5. Continue salt restriction.
6. Maintain suction on chest catheter if one is used—(10 cm. water).
7. Begin gastrostomy feedings early. (Total intravenous alimentation is extremely difficult in newborn).
8. Begin oral feedings third to eighth day. Determine time by amount of saliva aspirated by catheter.
9. If stricture develops pass string to aid in dilatations.

Common Pitfalls in the Management of Esophageal Atresia with Tracheo-Esophageal Fistula.

The following are common factors responsible for the increased mortality in these cases:

1. Suspecting the diagnosis late.
2. Passing the catheter blindly without the aid of fluoroscopy which may allow it to curl up in the blind pouch or pass through the trachea into the stomach by way of the tracheo-esophageal fistula.
3. Failure to aspirate the proximal stump, thus allowing further aspiration of mucous or oil once it is instilled.
4. The use of too much lipiodol, allowing some to spill over into the trachea.
5. The use of barium, not realizing its irritative properties, instead of lipiodol.
6. The failure to restrict saline intake, thereby causing peripheral and pulmonary edema.
7. Delay in operation hoping the baby will become stronger, or refusing to operate once pneumonia is present.
8. Failure to attempt a primary anastamosis or fanatical determination to perform one even in the presence of too much tension on the suture line.
9. Failure to consider the possibility of multiple anomalies.

DISCUSSION

Esophageal atresia with tracheo-esophageal fistula is one of the most serious congenital anomalies but it is amenable to surgical correction. Without repair this deformity is incompatible with life. One hundred per cent recoveries never will be possible because of the associated anomalies, prematurity, poor condition of some of the patients, aspiration pneumonia, and the all too frequent late establishment of diagnosis.

Penicillin has been an important adjunct in the lowering of the mortality but streptomycin has been found to be of even greater value as is demonstrated in the chart showing the marked lowering of mortality since 1947. Gastrostomy alone with feedings is worse than no operation at all since it increases the regurgitation through the fistula into the lungs.

Some men say that operation should not be performed in the presence of pneumonia. This statement is not true. If this were so, then surgical repair would be attempted on very few patients. Pneumonia exists, to some degree, in most of these cases after a few hours of extra-uterine life. Are there no means of combating pneumonia? Are we to condemn these

TABLE 3

Analysis of Cases

Swenson, Boston, Mass.
75 cases
57 Survivals (All end-to-end anastamoses except one in which the stomach was brought into the chest).
Leven, St. Paul, Minn.
77 Cases (1939–1950)
72 Cases operated (5 died soon after admission or permission for operation refused)
5 Cases—gastrostomy only.
67 Cases—Fistula closed and esophageal continuity reestablished.
47—Primary anastamosis
30—Survivals.
20—Multiple-stage procedures
11—Survivals.
19 Cases (1948–1950).
15 Cases—Survived.
Haight, Ann Arbor, Mich.
93 Cases (1939–1950)
86 Cases operated
41 Survived
3 Late deaths from unrelated conditions.
42 Cases (1947–1950)
29 Survived.
Abott, Atlanta, Ga.
22 Cases
15 Survived (all primary anastamoses).
Potts, Chicago, Ill.
32 Cases
2 Died soon after admission
1 Gastrostomy, died—presence of fistula not recognized.
29 Cases operated.
25 Primary Anastamoses
11 Survived
1 Late death.
Humphreys, New York, N. Y.
50 Cases
30–35 Per cent mortality.

patients to the certain death of drowning in their own secretions and withhold operation from them simply because they are not considered ideal candidates? Are we to delay operation for several days to "build up the patient" as is sometimes recommended? Of course, time must be allowed to establish fluid and electrolyte balance and to combat pneumonia, but

this period should be as short as possible and delay beyond this point is hazardous.

Early diagnosis is the single most important feature which will lower the mortality.

One should not hesitate to do multiple-stage procedures when primary anastamosis is not advisable. One should not risk a breakdown of the anastamosis in order to do a primary union if too much tension exists.

The results that have been obtained in several of the pediatric centers throughout the country are analyzed in the accompanying charts. These

TABLE 4

Analysis of Cases

	PRIMARY ANASTAMOSES	SURVIVALS
Leven	47	30
Potts	25	10
Abott	22	15
Swenson	75	57
Haight	86	44
Bigger	11	9
Total	266	165

TABLE 5

Cases Operated

1947–1950

	OPERATED	SURVIVALS
Bigger	7	7
Haight	42	29
Abott	20	15
Swenson	61	44
Leven	19	15

tables demonstrate what can be accomplished and should serve as a goal for many hospitals which, as yet, are unable to report a successful primary anastomosis.

SUMMARY

Esophageal atresia with tracheo-esophageal fistula is an uncommon congenital anomaly which is incompatible with life.

Satisfactory surgical correction has been perfected and offers the only chance for survival.

Early diagnosis is essential and methods for its establishment are outlined.

Mortality is high, but it can be reduced.

Pre- and post-operative management is discussed.

Surgical techniques are described.

Cases from several pediatric centers are reviewed.

REFERENCES

1. Turner, G., quoted by Ladd, W. E. and Swenson, O.: Ann. Surg. **125:** 123–140. 1947
2. Belsey, R. H. R. and Donnison, C. P.: Congenital Atresia of the Esophagus, Brit. Med. J. **2:** 324 (Part I), 1950.
3. Gibson, T., quoted by Bigger, I. A.: Ann. Surg. **129:** 5 (May) 1949.
4. Leven, N. L.: Congenital Atresia of the Esophagus with Tracheo-Esophageal Fistual, J. Thoracic Surg. **10:** 648, 1941.
5. Ladd, W. E.: The Surgical Treatment of Treacheo-Esophageal Fistula, New England J. Med. **230:** 625, 1944.
6. Haight, C. and Towsley, H. A.: Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula, S. G. O. **76:** 672, 1943.
7. Lyon, C. G. and Johnson, S. G.: Congenital Esophageal Atresia and Tracheo-esophageal Fistual, J. Thoracic Surg. **17:** 2, 162 (April) 1948.
8. Bigger, I. A.: The Treatment of Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula, Ann. Surg. **129:** 572, 1949.
9. Ravitch, M. M.: New Trends in Pediatric Surgery, Surg. Clinics. N. America, (October) 1949.
10. Swenson, O.: End-to-end Anastamosis of the Esophagus for Esophageal Atresia, Surgery, **22:** 324, 1947.
11. Potts, W. J.: Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula, J. Thoracic Surg. **20:** 5 (November) 1950.
12. Abott, O. A.: Personal Communication.
13. Reinhoff, W. F.: Bull. J. H. H. (January) 1948.
14. Haight, C.: Personal Communication.
15. Swenson, O.: Personal Communication.
16. Potts, W. J.: Congenital Deformities of the Esophagus, Surg. Clinics of N. America, (February) 1951.
17. Longmire, W. P.: Congenital Esophageal Atresia with Tracheo-esophageal Fistula, Arch. Surg **55:** 330, (September) 1947.
18. Pless, E. D.: Johns Hopkins Hospital Reports, **18:** 259, 1919.
19. Rienhoff, W. F.: Ante-Thoracal Transplantation of the Stomach in the Treatment of Congenital Atresia of the Thoracic Esophagus, Bull. J. H. H. **82:** 4, 496 (April) 1948.
20. Haight, C.: From Discussion quoted pp. 701–702, J. Thoracic Surg. **20:** 5 (November) 1950.
21. Leven, L.: From discussion quoted pp. 702–703, J. Thoracic Surg. **20:** 5 (November) 1950.
22. Humphreys, G. H.: From discussion quoted p. 703, J. Thoracic Surg. **20:** 5 (November) 1950.

CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: Richard J. Waters, M.D.
Sanford Leikin, M.D.
By Invitation: Bernice Wedum, M.D.
Richard J. Waters, M.D.

This four year old colored male was admitted to Children's Hospital on January 29th and died on February 10th.

He was well until two months before admission at which time he developed a sore throat, fever, and anorexia. He was seen by a local doctor and given a single injection of penicillin. Within four days the temperature was normal and he was once more indulging in vigorous outdoor activity with his playmates. Two weeks following this illness, he awoke crying and complaining of pain in the right hip. He remained in bed the rest of that day. The next morning he had a high fever and his nose bled twice during the day. The following day there was less fever and the pain had disappeared from the hip, but he complained of pain in the right knee, and his mother noticed some "puffiness" there. These symptoms disappeared, and the patient was active again within a few days. One month before admission the patient's mother noticed that he was irritable, inactive, and refused to play. Several days later, he was found crying, complaining of abdominal pain, and vomiting. A high fever developed and this continued for the next two weeks being accompanied by intermittent abdominal pain. He was seen by two physicians during this period and each prescribed tablets to combat "stomach gas." Two days before admission, a third physician felt an abdominal mass and advised hospitalization. On the day prior to admission, he was moderately "short of breath," and his mother noted swelling of the hands, feet, and eyes.

The family history revealed that the patient's father had rheumatic fever. It was not otherwise remarkable.

Past history revealed that the child was born normally at full term and that there were no neonatal complications. Prior to the present illness, there was no history of pharyngitis, nose bleed, chorea, or joint manifestations.

Physical examination on admission revealed a well developed but thin colored male in marked respiratory difficulty and who appeared acutely ill. There was no cyanosis or clubbing, but periorbital and dependent edema were marked. There was slight bulging of the anterior chest. The temperature was 99°, the pulse 150 per minute, the respirations 40 per minute, and the blood pressure 98/60. The tonsils were enlarged and injected. The

cardiac rhythm was described as "regular with a well marked gallop (diastolic) at the apex." A loud harsh systolic murmur was best heard at and inside the cardiac apex. There were crepitant rales over the bases of both lungs. The liver was palpable 6 centimeters below the right costal margin and ascites was present.

Fluoroscopy on admission revealed enlargement of the cardiac silhouette which filled the entire left chest. The right lung revealed diffuse infiltration which was interpreted as chronic passive congestion.

The patient was placed in oxygen and mercupurin was given intravenously. He was digitalized, the drug being given for the next four days. There was little improvement during this time. The patient gained two pounds in weight, the dyspnea continued, and the liver increased in size. The ventricular rate varied between 120 and 140 per minute, and the gallop rhythm persisted. On the third hospital day, a pericardial friction rub was heard. The apical systolic murmur became more harsh and was transmitted to both the left axilla and to the left back. Intravenous mercurials were again used, but the response was poor. On the fifth hospital day the temperature was elevated to 102°, and the fever continued at levels from 100° to 103° daily. Penicillin was administered for the remainder of the hospital stay. A subsequent x-ray revealed further cardiac enlargement and pulmonary congestion. The patient's condition had become critical and on the tenth hospital day cortisone was begun in 100 milligram daily dosage. Within twenty-four hours there was some decrease in temperature, however, the ventricular rate and respiratory rate continued to increase, and there was a progressive rise in blood pressure to 112/76. The patient expired on the thirteenth hospital day.

The following laboratory examinations were reported:

1. Urinalyses revealed good concentration in all specimens, 40–200 milligrams of albumin, no sugar or acetone, and on one occasion granular and hyaline casts were found.
2.

Blood Counts.	RBC	HGB	WBC	Granulocytes	Lymphocytes
1–29	3.5	9.0	17,300	58%	32%
2–2	3.4	8.9	11,800	40%	60%
2–7		9.4	10,000	63%	37%
2–9	3.2	9.0	16,000	77%	23%

3. Blood cultures were sterile.
4. Sedimentation rates were 34, 45, and 38 (all corrected).
5. No sickling was noted.
6. Eosinophile counts
   1st day cortisone— 1812
   2nd day cortisone— 578
   3rd day cortisone— 134

7. Blood chemistry:	
Blood chloride	479 mgs./100 ml.
Fasting blood sugar	174 mgs./ ml.
Non protein nitrogen	54 mgs./100 ml.
Total protein	5.89 gms./100 ml.
Albumin	3.07 gms./100 ml.
Globulin	2.82 gms./100 ml.
A/G. Ratio	1.1/1
8. Electrocardiographic study	
1. On admission	"P-R interval 0.2 sec., axis within normal limit, biphasic QRS complexes, first degree heart block."
2. After 43 hours and digitalization	"P-R interval .16, TI somewhat low, otherwise as previous tracing."
3. Four days after digitalis was discontinued	"P-R interval .14, T wave in lead 1 biphasic, and T wave in lead 3 appears notched."

DISCUSSION

Bernice Wedum, M.D.: This is an example of fulminating rheumatic pancarditis of approximately six weeks' duration in a four year old colored boy. It is not a problem in differential diagnosis. I am going to begin by discussing certain features of this case and conclude with a brief discussion of certain auscultatory findings in rheumatic heart disease.

An initial attack of rheumatic fever terminating fatally in six weeks is unusual. The disease is characterized by repeated episodes, and an attack predisposes an individual to subsequent attacks. A review of all cases of fatal rheumatic fever on file at the Army Institute of Pathology revealed one of four days' duration; the microscopic sections showed evidence of early Aschoff body formation although grossly the heart appeared entirely normal.

Rheumatic fever is more common among members of the colored race. This is not a matter of economic circumstance, but a true racial susceptibility. First attacks occur most frequently between the ages of five and nine years although one or two cases have been reported in infancy.

The manner of onset is classical with an initial pharyngitis—the so-called phase I, a period of apparent freedom from symptoms—phase II, and then the onset of rheumatic manifestations—phase III. However, only about 60 per cent of adults with rheumatic heart disease give a recognizable past history of rheumatic fever and when large groups of school children are examined, one-third to one-half of the cases found also give no past history.

Migratory joint pain is characteristic and the pain is severe. Actual visible swelling of the joints is more common in adults than in children. Nose bleeds are one of the minor manifestations of rheumatic fever. They

are usually scanty, occurring most often in the morning and are seldom found without other clear-cut manifestations of the active rheumatic state.

The course during the month preceding admission was one of progressive rheumatic carditis with the onset of congestive failure. Abdominal pain is itself a rheumatic manifestation but in this case it was probably due to the rapidly enlarging liver. Right upper quadrant pain is one of the very early manifestations of congestive failure in rheumatic carditis.

The familial incidence of rheumatic fever appears to be a genetically transmitted susceptibility. Experience with rheumatic fever among large groups of recruits revealed that only a certain proportion of these developed manifestations of the disease although environmental factors were constant. There are, however, other explanations which could account for this; one is that only a certain proportion of the individuals had been previously infected and sensitized to the streptococci prevalent and responsible for initiating the outbreak of rheumatic fever.

There is evidence that climate does influence the course and severity of the disease but not its prevalence.

The treatment of rheumatic carditis with congestive failure is a matter requiring careful clinical judgment, and one on which opinions differ. It is complicated by the fact that the use of certain drugs which are of therapeutic value in the active rheumatic state is debatable when far-advanced congestive failure is present because they are water retaining drugs—for example: sodium salicylate and cortisone. Salicylates when used should be given in the form of acetyl-salicylic acid. The use of digitalis is controversial. My own experience has been uniformly disappointing but the series of cases on which it is based is not large enough for positive statements. I prefer nasal oxygen; mercuzanthin, 1/10 milliter for every 10 pounds; and morphine, 1 milligram for every 10 pounds. I have had no experience with thromerin. A phlebotomy, a thorocentesis, and a pericardial tap are measures which should not be overlooked.

Granular and hyaline casts in the urine are the normal findings in congestive failure. Anemia is the rule in rheumatic fever and many children in the active rheumatic state over long periods develop an anemia which will not respond to iron. The white count is seldom markedly elevated. The sedimentation rate is almost invariably elevated in rheumatic fever although it may, on rare occasion, be normal. It often returns to normal with the onset of congestive failure.

The very high eosinophil count indicates that this child was making a very poor response to the stress of his illness. It would have been of interest to know what the total circulating eosinophil count was at the time of admission. I do not understand the high fasting blood sugar. An elevated non-protein nitrogen is the usual thing in congestive failure.

The decrease in the PR interval of the electrocardiogram in the course of digitalization is a variation in the course of the carditis and should not be interpreted as a result of digitalization.

Fernando R. Leyva, M.D.: Do you believe that there is a form of chorea distinct from the chorea of rheumatic fever or that all forms of chorea are rheumatic in origin?

Dr. Wedum: There is a group in Chicago which has published a large number of cases of chorea presumably non-rheumatic in origin. Chorea without an elevated sedimentation rate or evidence or carditis is clinically so indistinguishable from that occurring in the course of rheumatic fever that I have never found any reason to make a distinction.

Joseph M. LoPresti, M.D.: Is it not true that chorea is less frequently followed by rheumatic heart disease?

Dr. Wedum: That is true. The usual figure given is 25 per cent of the cases in which the initial manifestation is chorea compared with 50 per cent of the cases with polyarthritis as the initial manifestation.

Resident: Is chorea more common in females than in males?

Dr. Wedum: In our experience, chorea is only slightly more common in females than in males.

Resident: How long does it take for mitral stenosis to develop after the initial attack of rheumatic fever?

Dr. Wedum: It does not usually develop under two years although I have seen one case, I believe, in which we thought it had developed in over a year's time.

Recently in the course of observing a number of patients on whom commissurotomies were done for mitral stenosis, I have had an opportunity to confirm an impression which a number of us have that the murmur of mitral insufficiency is a very characteristic murmur and can be recognized and distinguished from all other apical systolic murmurs. It is of great importance that the nature of this murmur be clearly understood since it is the earliest manifestation of rheumatic heart disease in children. Children with this murmur should receive prophylactic penicillin while children who have only physiologic murmurs at the apex should not be labeled cardiac.

The murmur of mitral insufficiency is a long, blowing high-pitched systolic murmur varying in intensity from faint to loud heard best at the apex and transmitted into the axilla. This murmur usually increases in intensity after exercise as do most murmurs heard in children, and is heard best through the diaphragm of a Rieger-Bowles stethescope with the patient on the left side and holding the breath in expiration. This murmur is never transmitted to the neck. It is heard throughout systole right up to the second sound in contrast to most physiologic murmurs. It may sometimes

be louder and harsher in late than in early systole. This late harsh systolic component may be sometimes heard alone and probably indicates a mitral insufficiency which is gradually disappearing. All other systolic murmurs at the apex in the well child, with the exception of those which are loud and harsh and obviously organic, should be regarded as physiologic.

FIG. 1. Showing the opened heart as it appeared at autopsy. Note the thickened mitral valve and the verrucae along the line of closure on the auricular surface of the valve. The chordae tendineae are thickened and shortened. These features explain the insufficiency of the valve and the characteristic murmur on auscultation.

PATHOLOGIC DISCUSSION

Richard J. Waters, M.D.

We thank Doctor Wedum for being with us this afternoon and for her summary and comments pertaining to this case of rheumatic fever, rheumatic heart disease, and congestive heart failure.

At autopsy the heart of this patient presented the most significant pathological findings. The heart and pericardium were markedly enlarged

and occupied almost the entire width of the lower thorax. On opening the pericardial sac about 160 milliliters of clear straw-colored fluid escaped. The heart was three times the normal weight for this age group. The right atrium and ventricle were markedly dilated and their walls were thin. The interauricular and interventricular septa were intact. The leaflets of the tricuspid valve and the cusps of the pulmonary valve were normal although the valvular rings were dilated. The left atrium was slightly larger than normal. The mitral valvular ring was widely dilated. The mitral valve was thickened and the edges were rounded. The chordae tendineae were shortened and thickened. Along the line line of closure on the auricular surface of the mitral valve were seen numerous tiny, adherent, wart-like nodules (verrucae) which were gray, firm, and averaged one millimeter in diameter. The left ventricle was dilated and its wall was hypertrophied. The aortic ring was dilated but the valve cusps were normal. Histologic examination of sections of myocardium presented evidence of pan-carditis. The collaginous tissue appeared swollen and some fibrinoid degeneration was seen. This was particularly conspicuous about the arterioles. The endocardium in many areas was infiltrated by round cells and lymphocytes. Sections through the wart-like vegetations stained bright pink and revealed fibrin deposits in various stages of hyalinization. At the bases of the verrucae, fibroblasts and connective tissue fibers dipped into the underlying endocardium. The myocardial fibers were separated and between them were seen focal infiltrations of polymorphonuclear leukocytes and lymphocytes. The myocardial fibers were widened and cross-striations were absent. Around many of the arterioles in the myocardium were seen masses of hyalinized collagen. Within these masses were mononuclear cells, lymphocytes, polymorphonuclear leukocytes, and mesenchymal histiocytes. (Anitschkow myocytes).

The liver extended 7 centimeters below the right costal margin. It was dark purple and on cut-section revealed a typical "nutmeg" appearance as seen in passive vascular congestion of long standing. Microscopically, the hepatic sinusoids in the central zones of the hepatic lobules were dilated and filled with erythrocytes. There was pressure necrosis in the cords of hepatic cells in these areas. The lungs were twice the normal weight for this age group and were less crepitant than normal. The cut surface was beefy-red and bled freely. Histologic sections of lung tissue disclosed both pulmonary edema and marked passive congestion.

The spleen was also enlarged and presented features typical of vascular congestion.

The pathologic findings confirmed the clinical diagnosis and the cause of death in this patient may be attributed to acute pan-carditis resulting in marked pulmonary vascular congestion and edema.